Occlusive Auricular Thrombi

By John L. Read, M.D., Reno R. Porter, M.D., Simon Russi, M.D. and Joseph R. Kriz, M.D.

The literature on occlusive thrombi of the auricles has been reviewed in order to identify the symptom complex attributed to this condition. The clinicopathological findings are presented in four new cases which fit this symptom complex. Two cases are notable in that one is that of a ball thrombus occurring in the absence of mitral stenosis and another the first reported case of pedunculated thrombus of the right auricle.

ONE of the most interesting syndromes encountered in clinical medicine is that which develops subsequent to an occluding thrombus of the auricle. This is particularly true in the extremely rare case which involves the right auricle. The term “ball thrombus” was introduced by Wood, who reported the first case in 1814. In his now classic monograph published in 1924, Abramson made a complete survey of the literature. He reviewed 20 cases of ball thrombus of the heart and added one of his own. The first accepted case of occluding thrombus of the right auricle was reported by Wright and his coworkers in 1944.

Von Ziemssen, in 1890, originally suggested the clinical criteria for suspecting the diagnosis of ball-valve thrombus. In view of almost identical signs and symptoms occurring in his three reported cases, he suggested that the diagnosis of ball and pedunculated thrombi in the left auricle could be made in the presence of the following findings: (1) the presence of severe mitral stenosis associated with symptoms of extreme obstruction to the blood stream of the left heart, and (2) edema, coldness, and gangrene of the feet. He attributed the gangrene to thrombosis following extreme diminution in peripheral blood flow, less often to embolism. According to Abramson, most writers of that day looked upon the syndrome as a chance occurrence and, if not, one certainly compatible with a high grade of mitral stenosis and the endocarditis which so often accompanies it. Bozzolo, however, in 1896, focused attention upon Von Ziemssen’s criteria with a case diagnosed during life and elaborated upon Von Ziemssen’s clinical data. The former felt that the outstanding features of thrombosis of the left auricle were (1) signs of mitral stenosis, (2) signs of grave obstruction of the circulation to the left heart such as cyanosis, dyspnea, and cold extremities, (3) extreme feebleness of the peripheral pulses, and (4) the presence of many areas of gangrene in the lower extremities. The importance of the latter finding was reiterated by Redtenbacher, who stated that patients who lived long enough with cardiac thrombosis would inevitably develop gangrene of the extremities. Abramson emphasized the frequency of hemiplegia.

The popular sentiment at the turn of the century was that there was little in either the history or physical examination which might permit one to state that intracardiac thrombosis existed in a given patient with mitral stenosis. In 1909, Smithies appraised the clinical picture with the following observation: “It would seem to me, therefore, that the antemortem diagnosis of certain heart thrombi is not altogether impossible if the physical signs are carefully observed and recorded, and of these physical signs, I would lay especial stress on atypical peripheral vascular manifestations with more or less characteristic auscultatory and percussion signs in the heart itself. The manifestation of the embolic process should always furnish significant information.”

Although the symptom complex produced by massive auricular thrombosis is identical with that subsequent to a pedunculated or ball...
thrombus, the interest of most medical authors has centered almost exclusively about the select group designated “ball thrombi.” These must fulfill certain rigid criteria as defined by Welch; namely, there must be (1) entire absence of attachment with consequent free motility, (2) imprisonment in consequence of an excess in the diameter of the first narrowing in the circulatory passage ahead of it, and (3) such consistency and shape that the thrombus will not of necessity lodge as an embolus in passage. Obviously, as Aronstein and Neuman have observed, such a separation is exclusive but of academic interest since, clinically similar symptoms may be produced by a pedunculated thrombus and, as Yuskis and the authors have discovered, by a diffuse auricular thrombus. Of the 21 cases reported by Abramson, only two had been diagnosed during life.

Of the cases reviewed, 12 were females and 4 were males. The sex was not reported in five cases. The age was over 30 years in 12 cases with the most frequent decade being the fifth. In every case there was mitral stenosis of a marked degree. Since Abramson’s report, this diagnosis has been made with increasing frequency during life. The incidence of ball thrombi derived from several large series is roughly 1 in 2000 necropsies.

In 1934, Elson made one of the most significant contributions to the clinical diagnosis of ball thrombi. “The most important diagnostic feature, in our opinion, is the presence of comparatively rapid and transitory changes in the peripheral circulation, such as marked cyanosis or even gangrene which may involve the finger tips, toes, or tip of the nose. Cadaveric coldness, may occur suddenly, and quickly improve or disappear. The disappearance or diminution of pulsations, not from one extremity, but from several of them including both upper and lower, and their relatively rapid restoration should be emphasized. Such symptoms cannot be explained on the basis of peripheral emboli alone.”

In 1948, Evans and Benson attempted to evaluate the effects of the mass itself. Symptoms ascribed to 46 cases of ball thrombi reported up to that time were compared with those arising in 27 tumors of the left auricle. They found no significant difference in symptomatology. However, in 46 patients with mitral stenosis, who came to necropsy in the London hospital, only one patient who manifested chest pain as a significant feature during life failed to demonstrate an occlusive thrombus of the left auricle. Six of the 46 patients demonstrated occlusive thrombi. They therefore stress the importance of cardiac pain indistinguishable from that following coronary insufficiency. Unfortunately, four of their six patients with occlusive thrombi had no complaints of such pain during life.

A review of the available literature to date reveals approximately 60 reported cases of ball thrombi of the auricles and many more cases of massive and pedunculated thrombi.

**Case Reports**

During a six-month period in 1952, we were unusually fortunate at the McGuire Veterans Administration Hospital in seeing four cases of occlusive auricular thrombi, which we have considered to be especially significant from the clinical standpoint. The first case is rare in that it represents one of the few reported instances of ball thrombus of the left auricle occurring in the absence of mitral stenosis. The second case, and only one with rheumatic heart disease, is that of a massive thrombus lining almost the entire left auricle. The third is a case of triple thrombi of the left auricle attached loosely to the auricular wall in such a manner as to almost completely obliterate the auricular lumen. The fourth case is to our current knowledge, the first reported case of a pedunculated thrombus of the right auricle. Only two authenticated cases of ball thrombi of the right auricle have been reported to date.

Case 1. P. M., a 62 year-old Negro male, was admitted to the Surgical Service on Feb. 23, 1952, in a comatose state with gangrene of the right leg and evidence of peripheral vascular collapse. Subsequent history revealed a 40-pound weight loss and irregular heart action beginning two weeks previously followed by sudden pain in his right leg. He also noted recurring, intermittent abdominal cramps every few minutes of progressively increasing severity beginning 24 hours before. There
was persistent nausea, vomiting and passage of stools mixed with fresh blood. Past history revealed a history of hypertension of 12 years' duration, chronic alcoholism, and repeated urinary tract obstruction due to urethral stricture. There were four previous admissions for treatment of cardiac decompensation and urethral stricture. During these admissions he complained of dyspnea and epigastric pain and distension. His response to digitalis had always been adequate.

Physical examination at this time revealed a semicomatose Negro man in a state of considerable dehydration and inanition. He failed to respond to stimuli except to stir restlessly. The temperature was 96 F., respirations 22, pulse 150 beats per minute and blood pressure 100/80. The thorax was moderately emphysematous. The lungs were clear except for coarse breath sounds which were somewhat suppressed at the left base. The cardiac borders could not be defined satisfactorily by percussion. Heart sounds were heard best in the sixth left intercostal space along the sternum. The rhythm was rapid and grossly irregular. There were no definite murmurs; however, a distant whis-tling, rumbling sound was audible at the apex. This was felt to be probably of respiratory origin, but possibly represented an early mitral systolic or presystolic murmur. Examination of the extremities revealed bilateral femoral pulsations. The right lower leg was cold and revealed changes of early gangrene involving the right foot. There was a blotchy discoloration which extended to a point just below the knee where it coincided with the line of temperature demarcation. The right popliteal artery could not be palpated. There was 1 plus pitting edema present bilaterally. An electrocardiogram revealed auricular fibrillation with a rapid ventricular response (150) and long runs of ventricular tachycardia. He was given 1350 mg. of Pronestyl intravenously during the next six hours with complete disappearance of ventricular tachycardia and extra systoles. He was digitalized during the next 16 hours with 2 mg. of Cedilanid intravenously and intramuscular Digibid. His ventricular rate dropped to 90 beats per minute and he gradually regained consciousness on the afternoon of February 24.

Physical examination at this time, the day after admission, revealed exquisite tenderness and spasm of both upper abdominal quadrants. Examination of the heart and lungs was unchanged except that the previously described whistling sound could no longer be heard. That evening he again lapsed into coma. This was accompanied by increasing abdominal distension, vomiting, passage of tarry stools, fever and the appearance of an uremic frost on February 25. During this time the previously described murmur or respiratory sound again became audible. The night nurse noted that his pulse could often not be obtained at the wrist until the patient was turned on his side when it would come through quite well. He progressed rapidly into a uremic state and expired on Feb. 26, 1953.

Postmortem Examination: The heart weighed 900 Gm. There was a moderate dilatation, particularly of the auricles. Both ventricles were hypertrophied, the left ventricle measuring 2.5 cm. in thickness and the right 0.8 cm. An egg-shaped ball thrombus measuring approximately 5.0 by 3.5 cm. was found lying free within the left auricle (see fig. 1). The center was necrotic and yielded a pink-brown opaque fluid on puncture. All valves were normal. The coronary arteries were large and demonstrated no arteriosclerosis. The lungs were emphysematous and presented no other abnormalities.

Other pathologic findings included: an old myocardial infarction; thrombosis of the left popliteal artery with gangrene of the right lower leg; thrombosis of the coeliac artery; infarcts of the spleen, kidney and testicles; encephalomalacia; chronic hepatitis and a periurethral abscess.

Case 2. C. G., a 37 year-old white man, entered the hospital 48 hours after the sudden onset of severe

Figure 1. An egg-shaped ball thrombus measuring 5.0 by 3.5 cm. is seen lying within the chordae tendineae of the mitral valve. It is free of any attachments and has dropped from the left auricle during the process of cutting the heart. Note the marked left ventricular hypertrophy.
pain in his right chest, which was aggravated by respiration and accompanied by repeated bouts of hemoptysis. He was first seen at another hospital where physical examination demonstrated a pleural friction rub and a precordial thrill. The heart beat was rapid and grossly irregular. His temperature was 103 F. and there was a moderate leukocytosis. He had been started on penicillin and chloramphenicol therapy.

Chest roentgenogram revealed a greatly enlarged heart and a density in the left lung field which was interpreted as representing a pulmonary infarct. He gave a past history of rheumatic fever at age 14 and had apparently enjoyed good health until six months previously when he had been treated for a right middle lobe pneumonia and "heart trouble".

On admission, he presented a picture of collapse accompanied by mental confusion and marked dyspnea. He was moderately cyanotic about the face, lips and tips of his fingers and toes. His veins were collapsed. He complained of pain in his right side and repeatedly expectorated bright red blood. Examination of the chest revealed marked splinting, particularly on the right. There was dullness to percussion with medium moist rales elicited over both bases. No friction rub could be demonstrated.

The heart was greatly enlarged. The rhythm was grossly irregular with a rapid rate and a considerable pulse deficit. There was a precordial thrill and a questionable pericardial friction rub. There were no definite cardiac murmurs. Blood pressure was 80/60. Examination of the abdomen revealed liver dullness extending 2 to 3 cm. below the right costal margin. An electrocardiogram revealed auricular fibrillation with a ventricular response of 150, runs of ventricular tachycardia, and right ventricular strain.

The patient was treated with nasal oxygen, intravenous digitalization and Levophed in an attempt to combat his vascular collapse. He improved somewhat during the next six hours, but thereafter enacted an unusual and dramatic sequence of events. His clinical course was punctuated by repetitive episodes during which he would suddenly lapse into a state of profound collapse during which neither blood pressure nor pulse could be obtained. His attacks were invariably preceded by a period of irrational behavior, intense cyanosis about the face and lips, and inability to talk. This state was followed almost immediately by a generalized seizure lasting two or three minutes. Thereafter, a very rapid, irregular pulse gradually became apparent and the blood pressure once more could be obtained, although it never exceeded about 70/50 despite Levophed. His recovery became poorer and poorer following each seizure. After his fourth attack he failed to revive and expired 22 hours after admission.

The clinical diagnoses were: (1) Rheumatic heart disease with myocarditis, mitral stenosis and occlusive thrombus of the left auricle, and (2) pulmonary infarction.

Postmortem Examination: The heart weighed 850 Gm. and seemed to be unusually flabby. The left auricle and right ventricle were greatly dilated. There was slight dilatation of the left ventricle. The right ventricle was hypertrophied and measured 1 cm. in thickness. The left measured 1.5 cm. The left auricle was almost obliterated by a diffuse mural thrombus which varied in thickness from 0.5 to 1.3 cm. and lined almost the entire left auricular wall (see fig. 2). The mitral valve was stenotic and of a button-hole configuration. The remaining valves appeared normal. The coronary arteries were normal.

Microscopic examination was consistent with rheumatic myocarditis.

Other pathologic findings included: Edema and congestion of the lungs; infarction of the left lower lobe of the lung; chronic passive congestion of the liver and spleen, and pleural adhesions on the right.

Case 3. R. B., a 60 year-old, white man was admitted to the hospital on July 13, 1952, with a tentative diagnosis of acute coronary occlusion. A history was difficult to obtain because of profound
deafness and impaired cerebration; however, the pertinent facts were obtained in a letter from his family physician and from interviews with his wife. He had been treated during the previous 16 days for an arterial occlusion of his right lower leg, which had responded to conservative therapy. On the morning of admission, he suddenly developed crushing substernal pain followed immediately by deep cyanosis and unconsciousness. He gave a past history of three similar attacks during the previous year. Two of these had been associated with transient hemiplegia, which had resulted in some cumulative weakness. He had been completely disabled by his attacks since December 1951. At times, his seizures were preceded by a sharp, stabbing, epigastric pain. He had experienced an attack similar to the one seen on admission while enroute to the hospital.

Physical examination on admission revealed an elderly and markedly dyspneic man. His face and upper thorax presented a dusky, cyanotic hue. The retinal arterioles were the site of moderate arteriosclerosis. The chest appeared markedly emphysematous. Auscultation elicited many rhonchi and wheezes with moist basilar rales bilaterally. The heart was moderately enlarged to percussion. Heart sounds were quite distant with a sinus tachycardia (110 per minute). There were no definite murmurs or thrills. Blood pressure was 110/70. The abdomen was moderately distended and the liver edge was palpable 3 to 4 cm. below the right costal margin and moderately tender. Examination of the legs revealed a 2 plus edema of the right leg with impaired pulsations, some pallor and coolness as compared with the left.

Laboratory Data: Hemoglobin was 11.3 Gm., sedimentation rate 32 mm. in 1 hour, hematocrit 41, and white blood cells 11,350 per cubic millimeter with 81 per cent polymorphonuclear leukocytes. Urine contained 1 plus albumin. Wassermann test was positive. Blood urea nitrogen was 33 mg., and fasting blood sugar 87 mg. per 100 cc. Roentgenogram of the chest demonstrated a moderately enlarged heart with areas of possible pneumonia in the left upper lung field. The electrocardiogram revealed sinus tachycardia (rate 110), low voltage, and abnormal T waves.

A tentative diagnosis was made of arteriosclerotic heart disease and superimposed pulmonary disease with syncopal attacks on the basis of a Stokes-Adam's syndrome or occlusive auricular thrombosis. The patient required frequent aminophyllin and morphine.

He remained relatively comfortable until midnight on July 14, 1952, when he experienced a sudden smothering sensation accompanied by intense cyanosis, profuse icy perspiration and lapsed into a semicomatose state. During this period, neither pulse nor blood pressure could be obtained. Auscultation revealed marked pulmonary congestion. He was given intravenous morphine and Cedilanid with gradual improvement. On July 16, 1952, he was again seized with a smothering sensation. His pulse rate was 140 and very rapid and weak. The systolic pressure was 60 mm. Hg, the diastolic could not be obtained. Gross pulmonary edema developed. He appeared quite cyanotic and had marked distension of his neck veins.

On July 18, 1952, he began to cough up bright red blood. He continued to have almost daily paroxysms. On July 30, 1952, he suddenly grimaced as though in extreme pain, clutched his chest, became extremely cyanotic, gasped for breath and expired.

Postmortem Findings: The heart weighed 600 Gm. The left ventricle was slightly dilated with marked thinning and scarring. There was a large yellow area of necrosis with softening involving the entire posterior wall of the left ventricle. There were numerous almost confluent mural thrombi in both ventricles. The left auricle contained three rounded mural or pedunculated thrombi which met so as to practically occlude that chamber (see fig. 3). The
coronary arteries revealed marked atherosclerosis. The left ventricle varied in thickness from 0.7 to 1.5 cm.

Other pathologic findings include infarction of left lung, right pleural adhesions, fibrinous pleuritis on the left, generalized arteriosclerosis, and syphilitic aortitis.

Case 4. T. G., a 45-year-old white male, entered the hospital with a history of intermittent claudication in his legs of eight years duration. Six years previously he was told by a physician that no blood pressure could be obtained in his right arm, a condition which persisted for the remainder of his life. One year later he developed a transient left sided hemiplegia with residual fleeting paresthesias in all extremities and more frequent leg cramps after exertion. In May 1951, he was hospitalized for one month during which time he received anticoagulant therapy for a myocardial infarction. In September 1951, he noted the onset of daily fever, chills, and sweats. Because of these symptoms, he entered a University Hospital where he was found to have a positive tuberculin skin test and what appeared to be tuberculosis giant cells in his sternal marrow. His symptoms were alleviated by streptomycin therapy and he was discharged with a tentative diagnosis of miliary tuberculosis or hidden carcinoma. During the week preceding this admission he developed generalized ecchymosis with severe joint pains, particularly in his left hand. This member became cyanotic and the terminal phalanx of his left ring finger became gangrenous.

Physical examination on admission revealed a poorly nourished, chronically ill, white man. The temperature was 101 F. Pertinent findings were limited to the cardiovascular system, skin and extremities. The heart was of normal size and configuration. The rate was 110 beats per minute with a regular rhythm. There was a grade 2 apical systolic murmur. The blood pressure was 122/90 in the left arm, 140/78 in both legs, but could not be obtained in the right arm. Femoral and pedal pulsations were adequate. The abdomen was moderately distended with gas and the liver edge was palpated 3 fingerbreadths below the right costal margin. It was soft and moderately tender. The tips of all fingers of the left hand were cyanotic and tender. The tip of the little finger was black and dry. Examination of the skin revealed slopotty, brownish-red pigmented areas over the right wrist, both elbows, both hips and both thighs. Neurological findings were limited to generalized weakness with muscle wasting and anesthetia of pigmented areas.

Laboratory studies at the time of admission revealed an erythrocyte count of 3.5 million, hemoglobin 10.5 Gm. and sedimentation rate 32 mm in 1 hour. The leukocyte count was 9,800 with 64 per cent polymorphonuclear leukocytes, 10 per cent band forms, 19 per cent lymphocytes, 6 per cent eosinophils and 1 per cent basophils. The platelet count was 320,000, bleeding time was 1 minute and coagulation time 10 minutes. Blood urea nitrogen was 17 mg. per 100 cc and serum amylase 90 units (normal 0 to 60 units).

A tentative diagnosis of diffuse vascular disease, probably periarthritis nodosa, with subacute pancreatitis was made. Initial therapy consisted of vasodilator drugs, digitalis, penicillin and narcotics as required. A skin and muscle biopsy obtained shortly after admission was reported as consistent with thromboangiitis obliterans. The patient ran a febrile course with daily temperature elevations to 103 F. A stellate ganglion block on Nov. 26, 1951, produced dubious improvement and he was started on 25 mg. of corticotropin daily by eight-hour drip. This was discontinued on Dec. 20, 1951 because of his failure to improve. During this time, he complained bitterly of pain in his left hand and precordium. Repeated electrocardiograms revealed only low voltage and sinus tachycardia with occasional extrasystoles. On Dec. 23, 1951, he wakened with right facial weakness and inability to speak. During January 1952, he experienced repeated spells of air hunger accompanied by severe left precordial pain and cyanosis. During these attacks, his pulse became feeble and rapid with a drop in blood pressure to near shock levels. On two occasions he became comatose and no pulse or blood pressure could be obtained. Consciousness returned within a few minutes. His examiners were impressed by the absence of moisture in his lungs during these episodes; this was confirmed by roentgen examination. There was marked variation in the size of his liver, which varied from 1 to 6 fingerbreadths.

During February he became progressively weaker and his course was punctuated by more frequent spells accompanied by unconsciousness. The patient noticed that his attacks could be precipitated by moving from side to side in bed. He seemed most comfortable when lying curled up on his right side. On February 19, he developed frequent episodes of rapid grunting respiration associated with increasing cyanosis, which were followed by a sudden syncopeal attack from which he failed to recover.

Postmortem Findings: The heart weighed 325 Gm. Incision of the right auricle revealed a pedunculated thrombus which was attached to the endocardium of that chamber by a threadlike pedicle approximately 2 cm. in length. The thrombus was smooth, spherical and measured 3 cm. in diameter (see fig. 4). All valves were intact and normal. The coronary vessels were sclerotic with considerably narrowed lumens. Small mural thrombi were present in the apex of each ventricle. Infarcts were noted in the left lung, kidneys, spleen and left testicle. Histologic examination revealed two different processes involving the coronary arteries, one due to marked coronary atherosclerosis and the other to
OCCLUSIVE AURICULAR THROMBI

Fig. 4. The right auricle and ventricle are seen. A smooth, almost spherical thrombus, measuring 3 cm. in diameter, is suspended over the tricuspid valve by a slender pedicle. A smaller mural thrombus is seen in the apex of the right ventricle.

Thromboangiitis obliterans. The myocardium was the site of diffuse fibrosis and fatty degeneration. Changes due to thromboangiitis obliterans were noted in the heart, lungs, kidneys, pancreas, adrenals, extremities, spleen and testes.

Discussion of Cases

In retrospect, the diagnosis seemed to be fairly apparent in case 1. This, however, represented our initial experience with the syndrome and we failed to review the very important observations charted in the nursing notes concerning changes in the patient’s pulse with respect to postural variations. The whistling presystolic murmur was most unusual and no doubt became audible when the ball thrombus dropped into the mitral orifice, thereby simulating the effect of an extremely tight mitral stenosis. Changing murmurs are rarely observed although they have been stressed in the past by Battistini\(^{17}\) and others. Covey, Crook and Rodgers\(^{18}\) described a remarkable case in a 55-year-old woman who developed gangrene of the left leg concomitantly with the appearance of a loud presystolic murmur and precordial thrill. The murmur and thrill became progressively more pronounced, until finally the former could be heard a few feet from the patient. Evans and Benson\(^{16}\) noted that in cases of tumor of the left auricle, a systolic murmur was most often present, although a presystolic murmur was sometimes present. The fact that the murmur disappeared during our patient’s brief return to consciousness, only to recur when he lapsed back into coma, is considered especially significant by the authors.

A clinical diagnosis of occlusive thrombus was made in the second case. His repetitive episodes of peripheral vascular collapse, preceded by periods of irrational behavior, intense cyanosis about the face and lips, and inability to talk, strongly suggested the diagnosis. His profound heart failure, accelerated downhill course and shock-like state, which failed to respond to all therapeutic measures tended to reinforce the impression that an extraordinary lesion (occlusive thrombus) was present. Apart from the absence of gangrene, this patient’s findings were considered rather typical of the symptomatology displayed in an ideal case of occlusive thrombosis of the left auricle.

In the third case, it was our initial clinical opinion that the patient suffered with coronary heart disease and had perhaps suffered a recent myocardial infarction with the formation of a mural thrombus. In view of the duration of his symptoms, their repetitive nature (particularly with respect to the repeated attacks of transient hemiplegia), and the unusual clinical picture presented, it was also felt that he most probably had an occlusive thrombus of the auricle, possibly originating from the site of infarction of the left auricular wall.

The fourth patient was treated on another service and was seen by the cardiovascular department early in his course with respect to his generalized vascular disease. The majority, if not all of the occlusive phenomena, were most probably explained on the basis of
thromboangiitis obliterans. The syncopal attacks preceded by complaints of “air hunger”, clear lung fields, and marked variation in his liver size, fits exactly into the symptom complex of ball thrombus set forth by Wright and his colleagues.³

With the exception of case 4, the clinical picture presented by our patients was, in general, similar to those previously reported. In addition, we were especially impressed by the postural effects on the circulation manifested by the first and the last patient. Certainly such symptoms could not be explained on the basis of mitral stenosis per se although many of the symptoms once considered pathognomonic, such as intense cyanosis, particularly of the tip of the nose and extremities, can occur in the presence of mitral stenosis alone. We were also impressed by the occurrence of transient repetitive vascular phenomena initially limited to the central nervous system, as demonstrated by R. B., and of central nervous system manifestations preceding the onset of peripheral vascular changes, as presented by C. G. These may take the form of transient or permanent aphasia, syncopal attacks, dizziness, convulsive seizures, and hemiplegia. Usually, the central nervous system symptoms have been described as following the peripheral vascular symptoms, especially in the case of peripheral vascular collapse. Considering the vulnerability of cerebral tissue, it would seem logical to expect these symptoms to precede the peripheral symptoms. The fact that they usually do not, however, is probably related to the rapidity with which the systemic circulation is partially or completely obstructed by the thrombus. The symptom complex presented in these cases would appear to be reasonably definitive.

In view of the encouraging progress made in both the diagnosis and surgical alleviation of a variety of cardiovascular lesions, both congenital and acquired, it is probable that ultimately such thrombi may be demonstrated by angiocardiography and subsequently removed in the operating room concomitantly with a mitral valvotomy. It would appear that the first such step was taken by Bahnsen and Newman¹⁰ in 1952. They report a 54-year old woman, who when subjected to angiocardiography demonstrated a large filling defect in the right auricle. This defect proved to be produced by a large pedunculated myxoma attached to the fossa ovalis. The tumor was successfully removed at operation. The patient unfortunately succumbed 24 days later because of postoperative complications.

**Summary**

A review of the pertinent literature has been presented along with four new cases of occlusive auricular thrombi. Occlusive thrombi have been subdivided into (1) ball-valve thrombi, (2) pedunculated thrombi, and (3) massive thrombi. Patients with occlusive thrombi present a reasonably definitive symptom complex, manifested by peripheral and/or cerebral vascular manifestations, which are prone to be repetitive and variable. The murmurs occasionally wax and wane and all of the symptoms may be precipitated in some cases by postural variations. Two of the cases reported are notable in that one is the first reported case of pedunculated thrombus of the right auricle and the other is a case of ball thrombus occurring in a patient without mitral stenosis or rheumatic heart disease.

**Summario in Interlingua**

Es presentate un revista del pertinente litteratura insimul con quatro nove casos de occlusive thrombos auricular. Le thrombos occlusive es classificate como (1) thrombos globular, (2) thrombos pedunculate, e (3) thrombos massive. Patientes con thrombos occlusive presenta un satis definite complexo de symptomas apparente in peripheric e/o cerebral manifestaciones vascular que tende a repeter se e a variar. Le murmures aceresce e dekeres a vices, e omne le symptomas pote esser precipitate in aliam casos per cambiamentos de postura. Duo del casos in iste reporto es notable in tanto que le un es le prime unquam reportate de un thrombo pedunculate del auriculo dextere e que le altre es le caso de un thrombo globular in un patiente sin stenosis mitral e sin rheumatic morbo del corde.

**REFERENCES**

¹ Wood, W.: Letter enclosing the history and dissection of a case in which a foreign body was
found within the heart. Edinburgh M. J. 10: 50, 1814.


Occlusive Auricular Thrombi
JOHN L. READ, RENO R. PORTER, SIMON RUSSI and JOSEPH R. KRIZ

Circulation. 1955;12:250-258
doi: 10.1161/01.CIR.12.2.250
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1955 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/12/2/250

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/